

Management of Transient Cerebral Ischemic Attacks

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■ *Transient ischemic attacks (TIAs) are brief reversible episodes of neurological dysfunction due to temporary focal cerebral ischemia. Angiography should be performed only when operation is indicated or when the diagnosis is in doubt. Surgical treatment is recommended when the patient is a good surgical risk, when the stenosis is more than 70 per cent in the appropriate vessel and in certain patients with less severe stenotic lesions that appear to be a probable source of emboli. Anticoagulant therapy is indicated when there are recurrent TIAs, when the patient is not a good surgical candidate and when no appropriate surgically remediable lesion is found by angiography. If there is any significant contraindication to anticoagulants they should not be given. Discontinuance of anticoagulant therapy when the patient has been symptom-free for six months is recommended. In the experience of the authors the TIA syndrome is more benign in its course than was originally suspected and a conservative approach to surgical and anticoagulant therapy is recommended.*

IN THE WIDE SPECTRUM of strokes due to ischemic cerebral vascular disease, those manifest in the syndrome of transient cerebral ischemic attacks (TIAs) are the most challenging and provide the best therapeutic opportunities. In this report, current concepts of the pathophysiology, natural history and treatment of patients with TIAs are reviewed. Eleven case histories are included to illustrate the practical problems and decisions

involved in selecting the appropriate workup and treatment program for such patients. These cases are selected from a large series of patients from the Cerebral Vascular Research Clinic, Wadsworth Hospital, Los Angeles VA Center. During the last ten years, approximately 300 patients have presented with TIAs only.^{1,2,3} This constitutes about 10 per cent of all patients diagnosed as having ischemic strokes.

Transient cerebral ischemic attacks or transient strokes are brief, reversible episodes of neurological dysfunction due to temporary focal cerebral ischemia. Usually these episodes last only minutes to an hour, but they may occasionally persist as

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long as 24 hours. Frequently the attacks are recurrent, but it is not uncommon for a patient to have only one or two such attacks. Although vascular operations and anticoagulation may be useful in certain of these patients, the associated risks require that the clinician be judicious in the selection of patients to be so treated.

Pathophysiology

An awareness of some of the pathophysiological mechanisms underlying TIAs is important in evaluating and managing such patients. The primary abnormality common to all these patients is a transient decrease in delivery of blood to a focal area of cerebrum or brain stem. Many factors may be involved in the production of such focal ischemia. They may be categorized as local, regional and systemic. Locally the involved cerebral area may have a chronically marginal blood supply due to atherosclerotic stenosis of the major cerebral arteries or their branches or to sclerosis of arterioles directly supplying the area. A number of systemic abnormalities which may cause decompensation of such a partially ischemic area have been identified. These may include transient episodes of systemic hypotension, transitory cardiac dysrhythmia and other causes of diminished cardiac output, cerebral embolic events of cardiac origin, alterations of blood coagulation, hypoxia, hypoglycemia, polycythemia, anemia and even sudden rises in blood pressure with reactive vasospasm. Such systemic precipitating factors were identified in about 10 per cent of the TIA patients seen by us, an incidence that accords with the observations of Burrows and Marshall.⁶

Regional factors include atherosclerotic obstructive lesions within the neck vessels and, rarely, extravascular abnormalities, such as vessel kinking, fibrous bands and cervical spondylosis, which may cause intermittent mechanical obstruction of these vessels. These lesions may play a role in the production of focal cerebral ischemic phenomena, but direct proof of such a relationship is rarely available. While postmortem studies of patients with cerebral infarctions have shown a high incidence of extracranial vascular disease,^{11,13} similar changes have been noted in patients of comparable age without cerebral ischemia.²⁰ Extracranial vascular abnormalities are readily demonstrated radiographically and are frequently accessible to surgical intervention, but the clinical significance of such lesions in any individual case

is difficult to assess. Two angiographic studies of subjects without clinical evidence of cerebral ischemic disease uncovered extracranial arterial lesions in 23 per cent and 53 per cent of the subjects studied.^{5,9} On the other hand, many patients with TIAs have no demonstrable extracranial vascular lesions. Since extracranial arterial obstructive lesions may exist without symptoms of cerebral ischemia and symptoms of cerebral ischemia may occur in the absence of extracranial arterial occlusive disease, the coincidence of these two phenomena cannot be considered proof of a causal relationship. On the other hand, such a causal relationship undoubtedly exists in some symptomatic patients.

There are two ways by which extracranial obstructive lesions may cause cerebral ischemia. A decrease in the size of the lumen of the vessel may cause a reduction of blood flow distal to the obstruction. It is not commonly appreciated how severe a degree of obstruction is required to reduce blood flow. May, DeWeese and Rob,¹⁵ working with dogs, showed that an 80 per cent reduction of the lumen was necessary to reduce flow. Brice, Dowsett and Lowe⁴ demonstrated that carotid stenosis will significantly reduce blood flow only when its cross-sectional area is less than 2 square millimeters. Therefore, the presence of a stenotic lesion of an extracranial vessel, unless quite severe, would not be expected to decrease cerebral blood flow, locally or generally. The second way by which extracranial obstructive lesions may cause TIAs is by providing a source for emboli. In 1959 Fisher¹⁰ reported funduscopic observation of emboli passing through the retinal arteries during transient monocular blindness in patients with carotid artery disease. Honour and Russell¹² showed in rabbits that a series of experimentally produced platelet emboli commonly follow an identical course distally through the arterial tree. Thus, recurrent emboli might be expected to produce similar symptoms each time. Rough extracranial atherosclerotic plaques with only moderate stenosis may be a potential source of such emboli. Emboli may also arise from similar lesions of the larger intracranial vessels. The apparent salutary effect of anticoagulant therapy in many patients with TIAs is presumably due to a decrease in the frequency of such embolization as well as reduced sludging in low pressure collaterals.

Prognosis of TIA

Valid conclusions concerning the efficacy of therapy for a disease can be drawn only when there is adequate knowledge of the natural history of the disease. Since prolonged anticoagulant therapy and surgical relief of extracranial vessel obstruction are not without hazards, consideration of prognosis must influence decisions about individual patients. Only limited data concerning the natural history of patients with TIAs is available. Marshall¹⁴ reported that in a group of 61 patients presenting with recurrent TIAs who were followed an average of nearly four years, a major stroke developed in only one patient. These observations are similar to those in our own study.³ In a group of 30 TIA "control" patients followed up to eight years, only four had strokes; two of them recovered completely and two were left with mild residual effect. These studies suggest that the prognosis of this disease may be more benign than previously suspected, and this should be considered when decisions concerning therapy are made. Marshall¹⁴ reported another group of 68 patients who were seen initially with a major stroke which had been preceded by one or more TIAs. He was unable to ascertain any clinical features that would permit prediction of which patients with TIAs will eventually have major infarctions. Marshall's data on the two groups of patients should not be interpreted as evidence that over 50 per cent of patients with TIAs will eventually have cerebral infarctions. We believe that the difference between the two groups of patients is the result of the process of selection. The group of patients who present initially with TIAs alone may be viewed as similar to, say, a *prospective* study of patients with headache, who as a group have low morbidity and mortality and only rarely a brain tumor. Those presenting with a major stroke may be compared with a *retrospective* study of brain tumor cases, in which the history of headache is quite frequent. The actuarial expectation of strokes in persons who have had TIAs is as yet unknown. It is likely that many patients with one or few TIAs do not seek medical attention. Epidemiological surveys of large populations may be necessary in order to obtain this important information.

Management of TIA

The diagnosis of TIA may be clear-cut, but at times the differentiation between non-specific syncope, vestibular disorders, migraine, focal sei-

zures and TIA may be difficult. Cerebral neoplasms occasionally first come to notice through intermittent symptoms resembling those of TIA. In all patients a careful medical work-up should be performed. This should include careful auscultation of the neck for bruits and determination of the blood pressure and pulse in both arms. In certain cases significant systemic factors may be present. Hypertension, anemia, polycythemia, hypoglycemia or various forms of cardiac disturbance may be found. A reduction in cardiac output may be due to a developing myocardial infarction, angina, congestive heart failure or paroxysmal dysrhythmia. Since these conditions may themselves be responsible for the observed TIAs, treatment should be directed to the optimal management of them. Appropriate therapy for these conditions will often result in disappearance of the TIAs. Severe hypertension may also contribute to the production of TIAs. We believe that the carefully monitored gradual reduction of hypertension is an important part of management in these cases. We have seen a number of patients in which TIAs cease or decrease in number when hypertension is reduced. We do not use or recommend the use of vasodilators in the management of TIAs. There is insufficient evidence that they have value.

Indications for Angiography

Angiography is a valuable diagnostic aid in the management of patients with TIAs. However, since there is a small incidence of complications associated with this procedure, it should be carried out only when vascular operation is contemplated or when necessary to establish the diagnosis. In order to have adequate information to make decisions regarding operation, it is commonly necessary to visualize the neck vessels arising from the aortic arch. For detailed visualization of areas not seen on the study of the arch and for adequate intracranial arteriography, injection into individual vessels either percutaneously or via catheter may be necessary. A detailed discussion of the various applicable angiographic techniques and evaluation of angiographic findings is contained in Wilson's review article.²⁴

Surgical Treatment

Surgical treatment is at present available only for obstructive lesions within the extracranial vessels. The usual procedure is endarterectomy with

removal of atherosclerotic plaques with or without a patch graft to enlarge the lumen of the vessel. Various by-pass procedures are used under special circumstances. Attempts to reopen completely occluded vessels are generally unsuccessful and may be dangerous. We have found, as have others,²² that patients with evidence of severe chronic cerebral dysfunction or permanent focal motor deficit do not benefit from operation and run a higher risk of death or severe complications. At present we consider operation for stenosis of the extracranial vessels for TIA to be an elective procedure. Therefore, if a patient's general medical condition is poor, extracranial vessel operation should not be undertaken.

Vascular operation may be indicated in a patient with an accessible stenotic lesion of the carotid, innominate or subclavian arteries if the involved vessel is appropriate to the apparent location of the cerebral ischemia and if the stenosis is severe enough to cause reduction of distal blood flow. If the angiogram shows stenosis of 70 per cent or more, a reduction of that severity may be considered possible and operation may be recommended. Some investigators recommend operation for stenosis of appropriate arteries with as little stenosis as 30 per cent, based on the concept that such lesions may be the source of microemboli. Because anticoagulants may be equally effective and the surgical risks are significant, we prefer anticoagulation in this situation.

We believe that stenotic lesions of vessels not directly supplying the site of ischemia should not be operated upon if the appropriate vessel is free or relatively free of obstruction. Therefore we would not attempt to treat right cerebral hemisphere ischemia by operation on the left carotid artery, or to treat brain stem ischemia by operation on one of the carotid vessels if the vessel directly supplying the symptomatic area were relatively free of disease. On the other hand, the patient having right cerebral hemisphere ischemic attacks with angiographically demonstrated complete occlusion of the right carotid artery and severe stenosis of the left carotid artery, should be considered for left carotid operation. Such a patient has reduced collateral capability and is most apt to benefit from successful operation. Unfortunately his tenuous cerebral blood supply significantly increases the risk of the operation. Decisions in such cases are not easy to make and

require careful consideration of all pertinent factors.

Severely stenotic lesions of the origin of the vertebral arteries which are surgically accessible are seldom found and the surgical risks are greater. However, if both vertebrals are occluded, operation may be indicated for stenosis of the carotids which are providing crucial collateral supply. One other situation affecting the posterior circulation which is an indication for extracranial vessel operation is stenosis or complete occlusion of the subclavian or the innominate artery proximal to the origin of the vertebral artery, when this finding is associated with reversal of vertebral artery blood flow. This situation has been termed the "subclavian steal syndrome" and its classic clinical manifestations consist of brain stem TIA precipitated by exercise of the upper extremity on the involved side. It should be suspected in any patient with brain stem TIA and reduced pulses and blood pressure in one upper extremity.

Carotid artery stenosis is a fairly common incidental finding in patients who are completely free of symptoms of cerebral ischemia when arch studies are performed for other vascular disease problems. Likewise, asymptomatic older patients with bruits discovered over the carotids on routine physical examination commonly have carotid stenosis demonstrated by angiography. There are no data available which would support subjecting such asymptomatic patients to the risks of operation. McDowell and Ejrup¹⁶ recently reported on the benign course of patients with asymptomatic cervical bruits, confirming our own observations. If a patient with known carotid stenosis or bruit begins to have TIAs, then vascular operation or anticoagulants may be considered.

Surgical treatment has been advocated for excessive vessel kinking and for extravascular obstructive abnormalities such as anomalous fibrous bands and vertebral spondylotic spurs. We have found surgical repair indicated in only the rare case in which TIAs are precipitated by specific neck movements and where angiography confirms the transient, mechanical occlusion of the vessel.

Operations for relief of extracranial vascular obstruction entail significant risks. Because criteria for defining operative mortality and morbidity vary among reports in the literature, it is not easy to determine the incidence of complications. In addition, in many of the reports on surgical series,

patients with TIAs and those with cerebral infarctions with partial recovery are not clearly differentiated. Recent papers^{7,18,25} dealing with extracranial vessel operation in patients with TIAs report a surgical mortality of approximately 5 per cent. Another 5 per cent of patients have severe complications, such as cerebral hemorrhage or infarction, resulting in severe neurologic deficit. In our observation, these figures are optimal.

Anticoagulant Therapy

In our series the majority of patients with TIAs have not proved to be surgical candidates and only a few have had identifiable underlying systemic problems. Prolonged anticoagulant therapy may benefit these patients by reducing the incidence of platelet emboli arising from atherosclerotic plaques. Further, anticoagulants may prevent thrombus formation at the site of a stenosis and thus prevent additional reduction of distal blood flow. It is also possible that these drugs beneficially alter unknown blood coagulation factors or reduce sludging in low pressure collaterals which may have roles in the production of TIAs. Empirical clinical studies have shown that the incidence of TIAs is reduced by anticoagulants.^{8,17,19,21} There is also suggestive evidence that there is a reduction in the frequency of subsequent cerebral infarctions.^{8,21}

To avoid hemorrhagic complications, patients must be selected carefully. Liver disease, bleeding diathesis, active peptic ulcer and severe hypertension are contraindications to anticoagulants. If the patient has moderate hypertension which is readily controlled with medication, anticoagulants may be used but blood pressure should be monitored frequently. Impaired ability of the patient to cooperate, whether due to organic or psychological factors, may contraindicate the use of this therapy. We utilize warfarin and bishydroxycoumarin in the management of these patients. With careful management we have encountered no major and only a few minor hemorrhagic complications in long-term anticoagulant treatment of TIAs. Because the efficacy of anticoagulants in reducing significant morbidity among patients with TIAs remains uncertain, the use of these agents is not justified when even a relative contraindication exists.

The factors which are operative in the production of TIAs may change with time; for example, a source of emboli may disappear, a stenotic lesion

may occlude, collateral vessels may increase in size or number and coagulation factors may change. In our studies of the natural history of TIAs, we frequently observed that attacks may stop spontaneously. Therefore, lifelong administration of anticoagulants does not seem indicated. At present we recommend that treatment be maintained until the patient has been free of TIAs for at least six months. If TIAs have been reduced in number but have not disappeared, we would continue treatment. We have seen no significant rebound effect when anticoagulants are discontinued, but this phenomenon has been reported by other investigators, who recommend gradual withdrawal. If TIAs reappear after anticoagulant therapy is stopped, therapy should be started again.

Reports of Cases

The following cases are selected to illustrate some of the problems this disorder presents, the variable course of the TIA syndrome and recommendations for the management of these fascinating and challenging patients.

CASE 1.— The patient was a 66-year-old white man with a two-month history of frequent TIAs. These occurred one to two times a day, lasting for a few minutes. The episodes consisted of dizziness, diplopia, perioral tingling and quadriparesis. During the two weeks before admission to hospital, the episodes lasted up to 30 minutes. On the day of admission the patient had an episode of diplopia, left hemiparesis and numbness, which cleared in three hours. He had no significant past history. Results of general physical and neurological examinations were unremarkable. No bruits were heard and all pulses were normal. Blood pressure at the time of admission was 140/100 mm of mercury. During the stay in hospital the pressure was within normal limits. Laboratory studies including electroencephalogram, skull films and cerebral spinal fluid were normal. During the time in hospital the patient had several similar TIAs which were observed by physicians. The diagnosis was TIAs in vertebral basilar distribution.

An aortic arch study was performed in accordance with our research protocol. This showed no occlusive vascular lesions of the neck vessels. The patient was included in our anticoagulant study and was assigned, by random chance, to

the "treat with anticoagulants" category. He has been receiving anticoagulants for over two years and has had only two momentary attacks of diplopia and dizziness during that time.

Comment: We recommend that patients similar to this one with symptoms in the posterior circulation, should be treated with anticoagulants and the treatment continued until they have been free of symptoms at least six months. Because of the negligible discovery of lesions accessible to operation and the higher surgical risks involved, we do not believe angiographic studies are indicated.

CASE 2.—A 55-year-old white man, not hypertensive, experienced sudden onset of left facial weakness and dysarthria lasting 24 hours. The following day he had a second episode, very similar in nature, lasting several hours. He was admitted to hospital, where results of general physical and neurological examinations were unremarkable except for a soft bruit over the right carotid bifurcation. On the following day a right carotid angiogram showed stenosis of the right internal carotid artery intracranially in the region of the siphon. Immediately following right carotid angiography, the patient had an episode of left hemiparesis which cleared after four hours. As a part of our long-term anticoagulant study, this patient was, at random, designated to receive anticoagulant therapy. He has received anticoagulants for over four years and has had no further TIAs during this period.

Comment: At present we recommend an aortic arch study in cases of this type in which unilateral transient symptoms recur in the distribution of the carotid artery. Single vessel study, such as was done in this patient, involves more risks and might miss significant lesions in other vessels which could alter management. If the arch study showed only the carotid stenosis in the region of the siphon, which is inaccessible to operation, anticoagulant therapy should be used.

CASE 3.—An active normotensive 68-year-old white man with arteriosclerotic heart disease (three documented myocardial infarctions) had a ten-minute episode of vertigo while sitting in church. Twenty-four hours later he had a 45-minute episode of vertigo, blurred vision, paresthesias of the right extremities and weakness of the left extremities. This episode was not related to any specific movement or activity. The following day a similar 45-minute episode occurred.

He was admitted to hospital the next day. Noted on examination were blood pressure of 120/80 mm of mercury in both arms, full carotid and radial pulses on both sides, no bruits, no evidence of congestive failure or cardiac dysrhythmia, and normal neurological status. Results of routine laboratory studies including skull films, electroencephalogram and cerebral spinal fluid were within normal limits. An electrocardiogram was consistent with left bundle branch block. It was concluded that these episodes represented TIAs in the vertebral basilar distribution without clinical signs of a subclavian steal syndrome. This man was included in our study of anticoagulant therapy and, at random, was assigned to a control group. During follow-up over the next two years, with no specific therapy, this man had no further cerebral ischemic events.

Comment: This case illustrates the potentially benign course and unpredictability of this syndrome. We consider the history of significant cardiac disease in a man this age to be a contraindication for angiography and operation. This is particularly true when the symptoms are in the posterior circulation where not many operable lesions are found and the surgical risks are high. If there are no contraindications to anticoagulant therapy, this is the treatment of choice.

CASE 4.—A 59-year-old white man with emphysema, high blood pressure, arteriosclerotic heart disease and congestive heart failure, who had been taking digitalis, had two episodes of left hemiparesis and left hemisensory loss during a period of one week. One episode lasted 30 minutes and the other 15 hours. General physical examination confirmed the presence of the known diseases. There was a loud systolic bruit over the left subclavian artery, but good brachial and radial pulses were felt on the left. No significant abnormalities were noted in neurological and laboratory examinations. This patient was included in our anticoagulant study and was, at random, taken into the "treat" category. However, he changed his mind after this assignment had been made, and refused anticoagulants. During two years of follow-up he had three transient episodes of left hemiparesis, blurred vision and dizziness associated with exertion. Throughout follow-up the patient received digitalis, diuretics and a low salt diet.

Comment: Because of the seriousness of this man's other medical problems, we do not recom-

mend extracranial vessel operation. In cases in which operation is clearly contraindicated, there is no need to subject a patient to the small but significant risks of angiography. In patients with cardiac disease, especially those who have been in congestive heart failure or who have transient cardiac dysrhythmias, it is not uncommon to see cerebral ischemic symptoms secondary to diminished cardiac output.

CASE 5.—The patient, a 55-year-old white man who did not have hypertension, had ten episodes of left hemiparesis, lasting 15 to 30 minutes each, over a five-day period. These were not related to any specific activity. Results of general physical and neurological examinations were within normal limits. The blood pressure was 130/90 mm of mercury and no bruits were heard. Right carotid angiography revealed no abnormalities. This patient became, at random, a member of the control group in our anticoagulant study. During a three-year follow-up with no specific therapy, the patient had no further TIAs or other cerebral ischemic events.

Comment: Today we would perform an aortic arch study on such a patient. If no appropriate surgical lesions were found by arteriography, we would recommend anticoagulation.

CASE 6.—A 64-year-old retired ship's steward, was first seen because of complaint of recurrent 15-minute episodes of numbness of the left extremities and clumsiness of the left hand. He had had 10 or 15 such episodes during the preceding year. For three years, the patient had noted claudication of both lower extremities upon walking one block; and a year before the present examination he had had an aortographic examination at another hospital for evaluation of this problem. Bilateral iliofemoral operation had been proposed at that time but he refused. Hypertension had been present for six years and he was taking reserpine and chlorothiazide. General physical examination revealed bilateral carotid bruits and absence of femoral and distal pulses in both lower extremities. Results of neurological examination were within normal limits, as were routine laboratory studies, including an electroencephalogram, x-ray films of the skull and examination of the cerebral spinal fluid. An aortic arch study revealed severe narrowing of the right internal carotid artery to about 1 mm at its origin and mild (30 per cent) narrowing of the lumen of the left internal carotid at its origin. Pronounced obstruc-

tive disease of both common iliac arteries was also demonstrated. The patient elected not to have operation. He was then enrolled in the anticoagulant treatment study and was assigned at random to the anticoagulant treatment category. The patient has been treated with anticoagulants since December 1964 and has had no episodes of cerebral ischemia in that time. The patient has been receiving 0.5 gm of chlorothiazide daily and blood pressure has remained around 140/90 mm of mercury.

Comment: We recommend an aortic arch angiogram in this situation because of the clear-cut history of recurrent episodes of cerebral ischemia in the distribution of the right internal carotid artery plus the presence of bruits. The stenosis of the right internal carotid seen angiographically was severe enough to cause distal pressure reduction in this vessel, and therefore right carotid endarterectomy is recommended.

CASE 7.—A 63-year-old white man had five TIAs over a five-month period. Three of these episodes consisted of the sudden onset of dizziness and weakness of all four extremities, with complete clearing in one to two hours. Two of the episodes were restricted to sudden left hemiparesis which cleared within one hour. The patient was admitted to hospital and the general examination revealed blood pressure of 130/80 mm of mercury in both arms, normal retinal blood pressure and a systolic bruit over the right carotid bifurcation. Results of neurological examination and of routine laboratory studies including films of the skull, an electroencephalogram and cerebral spinal fluid examination were within normal limits. Right carotid angiography demonstrated severe (80 per cent) stenosis of the right internal carotid artery at its origin. It was our impression that this man had TIAs in the distribution of the vertebral basilar system and the right internal carotid system. In this case the patient was taken into our cerebral vascular disease anticoagulant therapy study, was assigned, by random chance, to a control group receiving placebos. During the first two years of follow-up the patient described only very occasional brief episodes of vertigo. During the next two years he reported frequent 15-minute to half-hour episodes of blurred vision, dysphagia and choking sensation. These episodes occurred as often as two or three times weekly. During the fourth year of follow-up he began to have occa-

sional five- to ten-minute episodes of visual loss in the right eye. Within three months he was having up to ten such episodes weekly and it was decided to begin anticoagulant therapy. Within two weeks after therapy was begun, the attacks of visual loss stopped and they have not returned. He has continued to receive anticoagulant therapy to the present (eight and a half years later), is working full time and is asymptomatic.

Comment: Today we would do an aortic arch study rather than simply a single carotid angiogram in such a patient. With demonstration of such severe stenosis, which very likely could cause distal blood flow reduction in the right internal carotid system, we would recommend endarterectomy. If operation were contraindicated for any reason or if the patient refused operation, anticoagulant therapy would be instituted.

CASE 8.—The patient was a 50-year-old white man with mild diabetes mellitus. On a routine yearly general physical examination, a bruit was heard over the right internal carotid artery for the first time. The remainder of the general and neurological examinations were within normal limits. No neurological symptoms were elicited. No specific evaluation or treatment was directed toward the bruit. During two years of follow-up the bruit has persisted, but the patient has remained asymptomatic.

Comment: Angiography is not recommended. We have found no evidence to support a surgical approach to an extracranial stenotic lesion in the absence of symptoms.

CASE 9.—A 51-year-old normotensive white construction laborer, while hanging curtains one evening, had sudden onset of tinnitus, tight feeling in the head, confusion, dysarthria, left-sided numbness and paresis and gait ataxia. This episode lasted six hours and then cleared completely. During the next three months he had approximately ten similar episodes lasting five minutes to an hour. Then he had an attack in which the left-sided numbness and weakness persisted for nearly 24 hours. He was admitted to hospital two days later and routine physical examination and neurological examinations revealed no significant abnormalities. Blood pressure was 125/80 mm of mercury in both arms and no bruits were heard over the neck. Results of routine laboratory studies including skull films, an electroencephalogram and cerebral spinal fluid examination were within

normal limits. The diagnosis was recurrent TIAs in the vertebral-basilar distribution. At the time we first saw this patient we were not performing aortic arch studies. The patient was taken, at random, into our cerebral vascular disease anticoagulant therapy study and was assigned to the control group. He has been observed for more than six years and during that time he has received no specific therapy. During follow-up he has had only occasional brief dizzy spells but recently he has had occasional attacks of angina. He works part time as a furniture assembler.

Comment: At present we would recommend an aortic arch study, to rule out a significant occlusive lesion of the posterior circulation accessible to operation. If angiography is not done or does not show a surgically remediable vascular situation, anticoagulant therapy should be begun. The possibility of a benign course without specific therapy is again illustrated by this case.

CASE 10.—A 69-year-old right-handed white man had experienced six episodes of right hemiparesis lasting five to ten minutes during the two months preceding hospitalization. Aphasia was present during one episode. Hypertension had been present for 15 years but the patient was not receiving therapy. There was no other significant medical history. Noted on general and neurological examinations were blood pressures of 190/100 mm of mercury in both arms, mild cardiomegaly, a loud bruit over the left carotid artery and no neurological findings. Laboratory work-up included an electrocardiogram interpreted as consistent with left ventricular hypertrophy and possible old posterior myocardial infarction. Aortic arch angiography showed complete occlusion of the right internal carotid and 90 per cent narrowing of the left internal carotid. Under general anesthesia a left carotid endarterectomy was performed without complication. The patient has been followed a year and a half and has been symptom-free.

Comment: Aortic arch angiography is indicated in this case because of the unilateral carotid distribution of symptoms. It is obvious that when one carotid artery is occluded, operation on the other carotid is more likely to result in a neurological complication than if only one vessel were involved. However, the reduced capacity for collateral flow makes relief of the partial obstruction more urgent and often worth the added surgical risk.

CASE 11.—The patient, a 63-year-old white man, suddenly became ill while standing in his living room. Vertigo, lightheadedness and visual blurring developed, with paresthesias in the left extremities, lasting a few minutes. Blood pressure was 180/90 mm of mercury in the right arm and 110/80 in the left. A bruit was heard over the right carotid artery and in the right supraclavicular region. The left radial pulse was weaker than the right and the right femoral pulse could not be felt. Aortic arch study revealed complete occlusion of the left subclavian artery and retrograde filling of the left vertebral and subclavian arteries. Bilateral carotid angiography showed pronounced diversion of the left common carotid artery flow to the distal left subclavian artery via the left external carotid artery collaterals. Left subclavian endarterectomy was performed without complications. On follow-up aortic arch study the left subclavian artery was patent. When the patient was last seen, blood pressure was 140/80 mm in both arms. He works full time as a salesman and has been symptom free for a year and a half.

Comment: In this situation aortic and carotid angiography is recommended because of the evidence for subclavian artery occlusive disease. Angiography demonstrated impairment of brain stem blood flow because of its diversion for retrograde subclavian filling. This is a relatively urgent indication for surgical intervention.

Discussion

In addition to the foregoing cases, the patient who presents with a single TIA deserves special consideration. Decisions concerning such a patient are not easy. In our experience cerebral infarction rarely occurs in patients who present with only a single TIA.²³ For this reason we do not routinely consider such patients candidates for vascular operation or for anticoagulants. Only if there is a significant cervical bruit or signs of subclavian artery obstruction do we proceed with angiography, operation or anticoagulation.

Another problem is the appearance of recurrent TIAs in a patient with a small fixed neurological deficit due to previous cerebral infarction. If the residual neurological signs are minimal, consideration for surgical or anticoagulation therapy would be the same as for the patient with TIA. A similar patient with more pronounced neurological residuum would be considered a poor surgical candidate. Our experience^{1,2} and other published

data⁸ are such that we do not at this time recommend anticoagulant therapy for the patient with a fixed neurological deficit (cerebral infarction) even though the deficit is not severe and a potential for worsening exists.

We have previously commented on the significant morbidity and mortality associated with surgical therapy. Angiography and anticoagulation also involve risks. It is therefore vital that such procedures be used only after careful assessment of all factors involved and with full knowledge of the many risks and the uncertain benefits.

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